Old muscle stem cells experimentally returned to youth

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Researchers at the University of California, Berkeley have found molecular pathways that human muscle stem cells rely on to repair damaged muscle. These pathways are active in younger people but less active in older people, explaining why muscles repair more slowly with age. The group found that younger volunteers had double the number of regenerative muscle stem cells in their thigh muscles compared to older volunteers. After two weeks in a leg cast, both groups began exercise routines to rebuild muscle. During this phase, the older volunteers had four times fewer muscle stem cells and rebuilt muscle more slowly. The researchers said that the poor response wasn't the fault of the older stem cells. Instead, signals in the aging muscle and blood locked the stem cells in an inactive state. From their work in mice, the researchers knew that proteins present in the muscle surrounding the stem cells helped these cells respond to distress signals from the injured tissue. In the human cells, they found a protein called MAPK that interprets these distress signals and triggers the muscle stem cells to begin the repair process. Young people have high levels of MAPK and older people have low levels of MAPK, providing one explanation for the older volunteers' poor response to exercise. In a lab dish, the group found that by artificially blocking MAPK in young muscle stem cells they could make young cells respond like older cells in a matter of days. The reverse was also true. Amplifying MAPK in older muscle stem cells in a lab dish rejuvenated the older cells. This work is an important step in verifying results from mouse stem cell aging studies in humans. The researchers hope their work could lead to therapies for muscle diseases and help older people to remain active, build stronger muscles and recover from injury.

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